

PERFUSION DEFECTS DETECTED BY THALLIUM-201 SCINTIGRAPHY IN CHAGAS' HEART DISEASE.

José A. Marin-Neto*, M.D., Paolo Marzullo*, M.D., Claudio Marcassa*, M.D., Lourenço Gallo Jr.*, M.D., Benedito C. Maciel*, M.D., Ricardo C. Bellina*, M.D., Antonio L' Abbate*, M.D., F.A.C.C., Medical School of Ribeirão Preto, Brazil* and CNR - Clinical Physiology, Pisa, Italy*.

Parasympathetic denervation, a unique hallmark of Chagas' Disease, may lead to sympathetic over-activity, thus causing ischemic myocardial lesions. We evaluated Thallium-201 myocardial scintigraphy in 23 chagasics, 18 men, aged 32-60 (mean = 42 years) who complained of atypical chest pain. Exercise was done on bicycle, with continuous 25 Watts incremental loads at 2min intervals, until profound dyspnea, fatigue or age predicted maximum heart rate occurred. Three-view scans were obtained immediately after maximum bicycle exercise and 3 hours later. None of the PTS had pain or ischemic changes on 12-lead exercise ECG. Semiquantitative analysis of 7 segments by three independent observers, blinded to clinical and laboratory data, showed perfusion defects in at least one segment, in all cases: 1-Reversible defects during effort in 6 PTS, 2-Fixed (effort and redistribution) defects in 16 PTS, mostly in apical segments. 3-Reversed redistribution defects in 15 PTS, mostly in posterior (10) and apical (5) regions. Resting wall motion abnormalities, detected by radionuclide or contrast ventriculography, correlated well with fixed but not paradoxical defects. Angiography in 16 PTS showed normal coronaries in all. We conclude that whilst fixed defects are likely to correspond to fibrotic lesions, the other defects may indicate the presence of regional flow/metabolism dysfunction possibly due to the autonomic impairment usually detected in Chagas' disease.

EVIDENCE OF CARDIOMYOPATHY IN SICKLE CELL ANEMIA: LEFT VENTRICULAR FILLING ABNORMALITIES IN ASYMPTOMATIC PATIENTS

Jannet F. Lewis, M.D., F.A.C.C., Barry J. Maron, M.D., F.A.C.C., Oswaldo L. Castro, M.D., Yunus A. Moosa, M.D. Howard University, Washington, D.C.

An underlying cardiomyopathy has been suspected (but not proven) in sickle cell anemia because patients often have symptoms of heart failure in the presence of intact systolic function. To determine whether left ventricular (LV) diastolic filling abnormalities exist prior to overt symptoms, Doppler-derived indexes of filling were obtained from the transmitral flow-velocity waveform in 30 consecutive asymptomatic patients with sickle cell anemia (age 18-39 years, mean 30) with normal systolic function and 30 normal subjects. Patients showed a spectrum of transmitral flow-velocity patterns - i.e., normal, impaired early filling and restrictive (rapid and abrupt) filling:

PATTERNS	No.	EF(m/s ²)	DCT(ms)	E(cm/s)	A(cm/s)
Impaired	7	3.4±0.6*	174±21*	57±11*	48±14
Restrictive	6	8.3±1.2#	108±11#	79±9	43±6
Normal	17	5.4±0.8	146±22	71±13	46±12

*p=0.02 (impaired vs. normal); #p=0.001 (restrictive vs. normal); EF=rate of decline of early velocity; DCT=deceleration time; E=peak early velocity; A=peak late velocity.

Thus, almost 50% of asymptomatic patients with sickle cell anemia and normal systolic function have subclinical evidence of abnormal LV filling, including a subgroup with a Doppler pattern suggesting restrictive cardiomyopathy. These abnormal patterns of LV filling may serve as early predictors of cardiac failure in sickle cell anemia.

Thursday, March 22, 1990

8:30AM-10:00AM, Room 36

Pediatric Cardiology: Interventional Catheterization Methods to Treat Congenital Heart Disease**TRANSCATHETER CLOSURE OF CONGENITAL VENTRICULAR SEPTAL DEFECTS**

Steve A. N. Goldstein M.D., Ph.D., Stanton B. Perry M.D., John F. Keane M.D., Jonathon Rome M.D., and James E. Lock M.D., F.A.C.C. The Children's Hospital, Boston, MA.

Between 3/1987 and 8/1989, 18 Pts were catheterized 20 times with intent to percutaneously close native or postoperative congenital ventricular septal defects (VSD). Indications were multiple episodes of endocarditis (n=1), shock (n=2), residual defects despite surgery (n=7), and planned surgery for congenital heart disease (CHD) requiring systemic ventriculotomy to close the VSD (n=9). The Rashkind double umbrella (12, 17mm) or Lock Clamshell occluder (17, 23, 28, 33mm) was used. VSDs were crossed via the IV to guide a venous catheter, long sheath and ultimately a device across the VSD from the right side.

In 4/18 Pts closure was not attempted: the VSD was <2mm (n=2), close to the Ao valve (n=1) or too large (n=1). VSDs in 14 Pts (0.7-44yr, 2-89kg) were 4-14mm in diameter and multiple muscular (n=7), single muscular (n=4), perimembranous (n=1) or patch margin (n=3). The device was placed "accurately" in all cases. One device was mal-positioned (6/8 arms on LV side of septum) and retrieved without intra-cardiac release; catheter VSD closure the next day was successful. All 15 released devices remained in stable position and abolished (n=13/15) or significantly reduced shunt through the VSD. Complications were femoral vein thrombosis (n=1), asymptomatic hemothorax (n=1), and umbrella impingement on septal leaflet of tricuspid valve (n=1, corrected by moving a device arm at surgery for CHD). Two Pts required general anesthesia; 1 developed post-extubation stridor. Follow-up (1wk-2yr) has revealed no other problems. Transcatheter VSD closure can be accomplished with limited morbidity and significant success as primary therapy or an adjunct to surgery.

OCCCLUSION OF PATENT DUCTUS ARTERIOSUS IN PIGLETS BY A DOUBLE DISK SELF ADJUSTABLE DEVICE

Eleftherios B. Sideris, M.D., Soula E. Sideris, R.N., Rani L. Ehly, R.T. Pediatric Cardiology, Amarillo, TX

To assess the efficacy and safety of a new patent ductus arteriosus (PDA) self adjustable double disk occluding device (SAD), 11 PDAs were occluded. The piglets were 10-15 days old and weighed 2.5-4.5 Kg. PDA was created by balloon angioplasty of the ductus in diameters up to 4mm. SADs were made of polyurethane foam disks with single floppy wire skeletons, connected together with an elastic thread. They were delivered transarterially through a 6F long sheath in sizes up to 12mm. The distal disk was released in the pulmonary artery and pulled against the ductus, while the proximal disk occluded the arterial side where it was automatically positioned and self adjusted for PDA shape and length. Follow-up right and left heart cath and angiography were performed up to a month from the occlusion and the piglets were sacrificed. All PDAs were occluded. There were no gradients across the occluders on either side. Endothelialization occurred in 2-3 wks. The optimal size of the device was 2.5 times the PDA diameter. SADs correct safely and efficiently PDAs of variable size and shape in piglets and could be applicable in humans, including infants.